tients. The call to organize a state medical society was issued by Dr. Thomas M. Logan, a South Carolinian, who settled in Sacramento in August of 1850, and Dr. E. S. Cooper of San Francisco, who came from Illinois in 1855. Realizing the need for unity in a medical organization, all individuals who called themselves doctors of medicine were invited to attend the meeting for the purpose of organization.

The status of the profession at that time, as described by an early historian, is of interest. Practitioners of the early 50's were said to consist of three parties: first, those who were earlier residents of California—"old-established practitioners"—and who were willing to have medical discussions, provided that only certain individuals were allowed to participate; second, a group smaller in number, composed of old-timers as well as newcomers—men who were anxious to see justice done to all, who had no animosities to settle, and were strongly bent on making the Society one of medical improvement; and third, a group composed of more recent arrivals in the state—leaders, active and progressive, but who were willing to make concessions for securing harmony. There was a violent conflict between the first and the third groups. The first "party" had been accustomed to habits of idleness, indolence and ease. Its members were more fond of amusement than of study, and could not brook the idea of being compelled to go to work in earnest for the advancement of medicine on this coast, or lose their claimed prestige in consequence of the system and activity of others.

Doctor Keene was selected to bring harmony among these conflicting groups. Because of his service in the State Senate he had become widely acquainted, and his reputation for honesty and integrity inspired confidence among all practitioners of medicine within the state. His untimely death might have forestalled the stormy existence of the Society during its early years, and its dissolution in 1860, not to be reorganized until October 19, 1870, when Dr. Thomas M. Logan, secretary of the newly established California State Board of Health, was elected president of the Medical Society. On that date, in his address before the Society, Doctor Logan said:

Prior to this organization, as most of you will remember, the medical mind was in a state of inertia—the profession in a chaotic condition. The dominant materialism of the Golden Age, which had invaded every department of human

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CLINICAL NOTES AND CASE REPORTS

UNDULANT FEVER (BRUCELLOSIS)

By GEORGE H. BECKER, M. D.
San Francisco

AS diagnostic procedures are improved, it would appear that Brucella infection is much more common in the United States than has heretofore been thought. Reports indicate that the known incidence of the disease has increased rapidly. The literature shows that much work is being prepared upon this subject. New techniques for diagnosing this condition are being brought forth from our research centers. The practitioner is now quite aware of the possibility in any case characterized by prolonged and wavering pyrexia.

Much less satisfactory are the reports upon therapy. So numerous are the treatments employed that the practitioner may yet be confused as to the best treatment to offer his particular patient.

In evaluating the reports upon therapy, we find that almost as many suggestions are offered as papers written!

Specific and nonspecific vaccines show some success. Various drugs and chemicals have been used with good results. Oral and intravenous methods are recommended. The series is usually small and many failures are noted; the one common factor in most successful treatments seems to depend upon a general systemic reaction or protein shock. The accompanying fever has usually been transient and more or less uncontrollable.

Regardless of methods used, the best results seem to follow the more severe thermal reaction. Most writers agree that the specificity of vaccines used is unimportant, and the results vary chiefly with the degree and extent of the reaction. As some patients respond but slightly to vaccine injections, it would seem logical to produce the hyperpyrexia by more easily controllable means.

Artificial fever can be easily regulated, both as to degree and duration, by one of the several mechanical methods now in use.

The following is an account of the treatment of a case of brucellosis by artificial fever, followed by permanent recovery.

REPORT OF CASE

Mr. J. A., aged 57, executive-salesman. Admitted to hospital on January 31, 1938. Present illness began with loss of appetite and weight, lethargy, lack of ambition, weakness, slight cough, gastro-intestinal distress, daily fever. This was about Christmas Day, 1937.

A diagnosis of influenza was made and the patient confined to bed and treated with salicylates and forced fluids. After three weeks of such treatment he was seen by the writer.

The history showed that, as an executive, he had no contact with any animals, and, disliking milk, he had only occasionally consumed milk, which was always pasteurized. Questioning, however, revealed week-end visits to a friend's country place, upon which were some cows and hogs. On these visits he occasionally participated in work with the animals and about the barns. He also recalled that, while deer hunting, he drank water carried from the dairy in five-gallon milk cans.

Past History.—His health had always been excellent, except for a gastric ulcer twenty years ago, which was healed by medical treatments. No operations or accidents.

Examination showed a man of fifty-seven years lying in bed quite depressed and apprehensive. He had lost about thirty pounds weight (from 143 to 112 pounds), and presented an emaciated appearance. The temperature was 101.4 degrees Fahrenheit; the pulse, 120; respiration, 26; the blood pressure was 125 systolic, 80 diastolic. The head and neck presented no abnormalities, save small atrophied tonsils. The thyroid was slightly palpable. The heart was negative, except for a soft systolic murmur at the apex, not transmitted. A few coarse bronchial râles were present. The abdomen was negative to examination, as were the extremities. The reflexes were all present and physiologic.

Laboratory Examination.—Blood count: Hemoglobin, 14.7 grams; red blood cells, 4,820,000; white blood cells,

10,800; neutrophils, 24 per cent; lymphocytes, 68 per cent; monocytes, 5 per cent; basophils, 1 per cent; eosinophils, 1 per cent. Urine was negative for albumin, sugar, and abnormal cells. Widal was negative. Wassermann was negative. Roentgenograms of the chest were negative. Undulant fever agglutination was four plus in dilutions from 1:40 to 1:2560, for Brucella abortus. Phagocytic index for Brucella, 18.16. Complement-fixation for Brucella, positive in 0.0005 cubic centimeters (four plus). Absorption test, abortus-porcine group.

Following the diagnosis of undulant fever on January 19, 1938, sulfanilamide therapy was instituted, and the patient received 30 grains the first day and 60 grains daily for the following ten days. The symptoms were affected but slightly. The patient was hospitalized on January 31, 1938, and vaccine therapy instituted.

Mixed typhoid vaccine was given intravenously and repeated every two to three days in a dose of seventy-five million. The usual reaction of chill, fever (103.4 degrees), sweats was obtained with each injection. Within twenty-four hours the temperature was normal and remained so until the next injection. These were repeated every two to three days for six treatments, after which the patient was afebrile for two weeks.

With a return of the fever we instituted artificial fever therapy. The temperature was elevated to 103.8 degrees Fahrenheit and maintained for five hours, following which temperature returned to normal. He was dismissed from the hospital on March 8, 1938. His appetite returned, his weight improved, and within a few weeks he was back at work and has since enjoyed the best health of his life, with a steady increase in weight. He is now weighing his maximum (150 pounds) one year later.

COMMENT

We offer a case which failed to respond to the commonest forms of therapy, and was permanently cured by artificial fever therapy.

450 Sutter Street.

EXTREME LEUCOPENIA IN CHRONIC PLEURAL TUBERCULOSIS

By PHILIP KING BROWN, M. D.
San Francisco

REPORT OF CASE

C. D., age sixty-four years, employed as a pumper; twenty-five years' service.

When he entered the Southern Pacific Hospital on December 14, 1935, after having had three or four entries for minor injuries (the latest one nine years previously), he was found to have an acute bronchitis, possibly an influenza, which was epidemic at the time, with a pleuritis at both apices. His pulse was between 80 and 100 when he entered. He had no fever. His blood pressure, however, was 170/98. No findings in the urine. He had a red count of 4,000,000; 76 per cent hemoglobin; 3,200 whites; 78 per cent neutrophils; 17 lymphocytes; 5 large mononuclears.

His x-ray report showed discrete calcic deposits in the upper portion of the left-lung field and near the left base, with an irregularity of the right diaphragm; clouding of both apices.

He made an uneventful recovery.

He reëntered on January 8, 1936, with a fracture of the left humerus. He ran a low fever of one-half to one degree for four weeks, and his pulse was constantly rapid. He was discharged from the hospital with the fracture repaired, but still with a rapid heart and one-half degree of fever. His blood counts were significant: 69 per cent hemoglobin, 3,500,000 reds on January 9; increased to 91 per cent hemoglobin, and 4,500,000 twenty days later:

1936	White Blood Cells	Neutrophils	Lymphocytes	Large Monocytes
January 9	1,900	72	26	2
January 10	2,250	74	22	4
January 15	1,750	72	22	6
January 29	1,000	70	24	6

The patient again reëntered in January, 1937, with a bronchitis and a very rapid heart, between 90 and 102. The significant factor this time also was the leukopenia with 3,250 whites, 45 per cent neutrophils, 50 lymphocytes, and 4 large monocytes, with one per cent eosinophils.

Two months later he was back in the hospital with his now chronic bronchitis, and again a very rapid heart action and a hypertension of 180/100. There was no anemia, but 1,550 leukocytes with 52 per cent neutrophils, 44 lymphocytes, 2 large monocytes, and 2 eosinophils.

He was pensioned and took good care of himself, so he said, but returned to the hospital in August, 1938, and remained until his death, a month later. His heart fibrillated all this time, and there was a decompensation and a pleurisy with effusion on the right side. He ran a fever to 100. His pulse rate at the wrist was recorded between 90 and 120. He refused to have fluid withdrawn from the chest and refused a sternal puncture in order that we might make some study of the hemopoietic system.

He entered with a slight anemia, which improved with iron. The significant thing, however, was his marked leukopenia with relative lymphocytosis. The counts were as follows:

1938	White Blood Cells	Neutrophils	Lymphocytes	Large Monocytes	Eosinophils
August 23	750	48	52	....	....
August 24	800	64	24	12	....
August 30	1,150	56	42	....	2
September 1	500	72	28	....	....
September 6	600	28	72	....	....
September 9	750	36	60	4	....
September 13	350	40	56	4	....
September 16	250	40	60	....	....
September 19	1,500	64	28	8	....

The patient was followed in the out-patient department on several occasions when not in the hospital, and various remedies were used which we thought might increase his leukocyte count. It is interesting to note that, on several occasions, the blood pressure was found to vary between 138/78 and 170/110.

The tendency to an anemia was treated constantly with iron, and various methods of stimulating the white cells were tried—sterile milk among them. The white count in October, 1936, was 1,600 with 60 per cent lymphocytes, and this occurred one year after he had had a pneumonia, presumably influenza. The neutrophils got up to 56 on one occasion and 64 on another, but the total whites did not rise above 1,400. At a later period, when he seemed quite well, his total whites were 1,750 with 76 per cent neutrophils; but on the last out-patient visit before his final entry to the hospital he had only 750 cells with 30 per cent neutrophils.

Autopsy No. 975A. September 20, 1938. A. M. Moody, M. D., Pathologist.

Cause of Death.—Tuberculous pleuritis with effusion, compression atelectasis and pulmonary thrombosis with infarction, and terminal lobular pneumonia.

Gross Anatomy.—The spleen is 15 by 11 by 4 centimeters and weighs 335 grams. It is hyperplastic in type and its capsule is irregularly thickened.

The gall-bladder contains thick bile and seven gallstones, the largest of which is 1.2 centimeter in its greatest diameter.

The external surface of the right lung is covered with a fibrinopurulent exudate over the inferior portion of the upper lobe, and the entire surface of the middle and lower lobe. There is a compression atelectasis and some consolidation of the lower lobe. There is an embolic-like occlusion of the pulmonary artery to the lower lobe.

Histologic Examination.—Lungs: Fibrinocaseous pleuritis (tuberculous), compression atelectasis, edema, pulmonary thrombosis and infarction with terminal lobular pneumonia.

Heart: Moderate coronary arteriosclerosis, myocardial hypertrophy, and edema.

Liver: Cloudy swelling and slight fatty changes.

Gall-bladder: Chronic cholecystitis and cholelithiasis.

Adrenals and pancreas: Unaltered.

Spleen: Hyperplastic splenitis with chronic (perisplenitis) thickened capsule.

Kidney: Scattered regions of interstitial nephritis, moderate edema and cloudy swelling.

Urinary bladder: Unaltered.

Bone marrow: Devoid of noteworthy alterations.